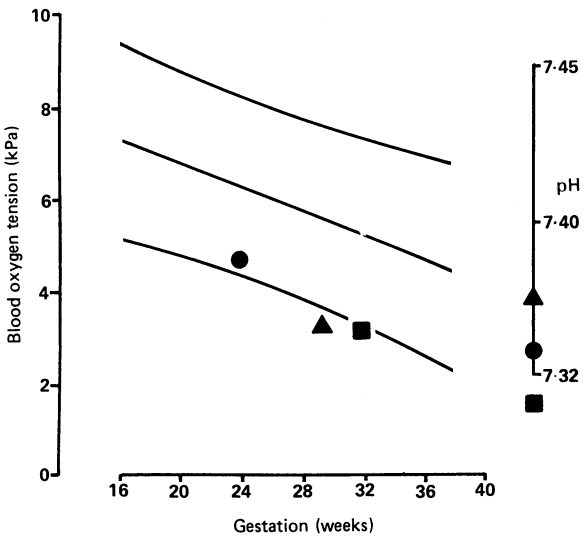
and 5 days' gestation considerable spontaneous decelerations in the fetal heart rate were noted. Further analysis of cord blood showed the fetus to be acidotic (pH 7·31) and hypoxic (oxygen tension 3·3 kPa), and a 1660 g female infant was delivered by caesarean section. The infant required intubation at birth (Apgar scores were three at one minute, seven at five minutes; cord blood pH 7·20) and was transferred to the neonatal intensive care unit. No complications occurred during the postnatal period, and the baby was discharged after 22 days.



Oxygen tension and corresponding pH in cord blood plotted against normal ranges (mean and 95% confidence intervals) during gestation.

Comment

The traditional assessments of fetal growth and activity did not show hypoxia whereas the routine Doppler studies of blood flow allowed the condition to be identified and hence allowed the fetus to be delivered successfully before death could occur. Interestingly, fetal growth recorded by ultrasonography continued normally; abnormalities in blood flow and chronic fetal hypoxia are usually associated with fetuses that are small for gestational age.[5] Owing to the suboptimal control of diabetes in the mother, as shown by the high concentrations of glycosylated haemoglobin, the fetus might have been expected to exceed the average birth weight for gestational age. Most infants born to our diabetic patients (79%) weigh more than the 50th centile. The birth weight of this infant was still on the 35th centile despite chronic hypoxia in utero, suggesting that, although the infant was of an appropriate weight for gestation, she was growth retarded and the accelerating effects of maternal diabetes on growth had been counterbalanced by "placental insufficiency."

Analysis of fetal blood gas tensions at the time of delivery suggested that some infants of diabetic mothers are fairly hypoxic.[1] We recently showed by cordocentesis that the blood oxygen tension of such infants in utero is much lower than normal, providing an explanation for previously "unexplained" late fetal death. Doppler studies of blood flow and cordocentesis may allow fetal hypoxia to be detected earlier than the traditional tests of fetal wellbeing.

1 MacFarlane CM, Tsakalakos N. Evidence of hyperinsulinaemia and hypoxaemia in the cord blood of neonates born to mothers with gestational diabetes. *S Afr Med J* 1985;67:81-4.
2 Nicolaides KH, Soothill PW, Rodeck CH, Campbell S. Ultrasound guided sampling of umbilical cord and placental blood to assess fetal wellbeing. *Lancet* 1986;i:1065-7.
3 Soothill PW, Nicolaides KH, Rodeck CH, Campbell S. The effect of gestational age on blood gas and acid-base values in human pregnancy. *Fetal Therapy* 1986;1:166-73.
4 Nicolaides KH, Bilardo CM, Soothill PW, Sel T, Campbell S. Absence of end diastolic frequencies in the umbilical artery as a sign of fetal hypoxia and acidosis. *Br Med J* (in press).
5 Soothill PW, Nicolaides KH, Bilardo CM, Campbell S. Relation of fetal hypoxia in growth retardation to mean blood velocity in the fetal aorta. *Lancet* 1986;i:1065-7.

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Lactose absorption and milk drinking habits in Estonians with myocardial infarction

In 1980 Segall suggested that a diet rich in lactose might be a risk factor for ischaemic heart disease.[1] Data from 29 different ethnic groups were compared, and a correlation was found between mortality from myocardial infarction on the one hand and efficient lactose absorption in adults and the consumption of milk on the other. Consumption of large quantities of milk is facilitated by high intestinal lactase activity, which is an inherited characteristic.[2]

We undertook a case-control study to test whether the consumption of large quantities of milk and the unhindered absorption of lactose increase the risk of myocardial infarction. The incidence of myocardial infarction in Estonia is one of the highest in the Soviet Union, and the prevalence of primary adult type lactose malabsorption is 24-32%.[2]

Subjects, methods, and results

We studied 70 Estonian men aged 36-78 who were admitted to hospital with acute myocardial infarction. A male patient was matched to each case for age, nationality, and place of residence (urban/rural). These controls had no ischaemic heart disease but were in hospital because of trauma or otorhinolaryngological disease. Informed consent was obtained from each patient. Lactose absorption was determined by a standard 50 g lactose load four weeks later. When the rise in blood glucose concentration was less than 1·1 mmol/l the patient was considered to have lactose malabsorption. There were no cases of general malabsorption as shown by a glucose-galactose load. All the subjects were asked what their average daily regular consumption of fresh milk (200 ml glasses) had been before they became ill.

We used the χ^2 test to calculate the significance of the difference in milk consumption and the prevalence of lactose malabsorption between the two groups. The relative risk of myocardial infarction was expressed by odds ratios comparing differences between the case-control pairs whose consumption of milk differed.[3] Several potentially confounding factors (hypertension, family history of ischaemic heart disease, cigarette smoking, and overweight) were also considered in multiple logistic regression models. Corresponding odds ratios and 95% confidence intervals were calculated from the coefficients and their standard errors.[3,4]

The regular milk consumption in men who had had a myocardial infarction exceeded that of their controls (table; $\chi^2=14·80$, $p<0·01$). The relative risk of myocardial infarction in those drinking three glasses of milk or more daily was four times that of those who drank less than three glasses. When adjusted for potential confounding factors the estimated relative risk for drinking three glasses of milk or more compared with drinking less than three glasses daily was 1·7 ($p<0·05$).

Association between milk drinking and myocardial infarction

Regular milk consumption (No of glasses daily)	Men with myocardial infarction (n=70)	Control subjects (n=70)	Estimated odds ratio (95% confidence interval)
<3	38	59	1·0*
≥3	32	11	4·0 (1·4 to 13·3) 1·7† (1·1 to 2·6)†

*Reference value.
†Figures adjusted to allow for confounding variables.

Twelve of the patients with myocardial infarction and 22 of the controls had lactose malabsorption ($\chi^2=3·88$, $p<0·05$). The estimated relative risk for lactose absorbers and malabsorbers was not significantly different from 1·0.

Comment

Although lactose malabsorption was less common in the patients than the controls, the calculated relative risk failed to show a significant association between myocardial infarction and lactose absorption. Drinking milk in large quantities, however, significantly increased the risk of infarction. The risk associated with milk drinking was independent of hypertension, overweight, cigarette smoking, and a family history of ischaemic heart disease.

Lactose absorption does not seem to be a risk factor for myocardial infarction by itself but is a precondition for the ability to drink a lot of milk without getting complaints. With that proviso our results support the hypothesis that there is an association between ischaemic heart disease and milk intake.[1]

Before any major dietary changes can be recommended it remains to be clarified which component(s) of milk are responsible. None the less, it seems advisable for adults to drink milk only in moderate quantities.

We thank Professor K Villako for the idea of the study and associate professors M Uusküla for his help in collecting the material, T Sahi for advice and reading the manuscript, and L-M Tooding for statistical analysis.

1 Segall JJ. Hypothesis: is lactose a dietary risk factor for ischaemic heart disease? *Int J Epidemiol* 1980;9:271-6.
2 Isokoski M, Sahi T, Villako K, Tamm A. Epidemiology and genetics of lactose malabsorption. *Ann Clin Res* 1981;13:164-8.
3 Schlesselmann JJ. *Case-control studies. Design, conduct, analysis*. Oxford: Oxford University Press, 1982:354.
4 Engelman L. Stepwise logistic regression. In: Dixon WJ, *et al*, eds. *BMDP statistical software*. Berkeley: University of California Press, 1981:330-41.

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Implications of two newly opened ice rinks on an accident and emergency department

Previous studies have reported many injuries sustained at ice rinks.[1,2] I report a study prompted by the almost simultaneous opening of a 60×30 m Olympic rink, currently the largest in the United Kingdom, and a 38×22 m "family fun rink" in the catchment area of this hospital.

Methods and results

Injuries treated at this department during the six months after the rinks were opened were monitored to assess the number and nature of injuries resulting from skating, the cost of treating them, and the measures that might be taken to make skating safer.

A total of 636 patients (268 male and 368 female) presented to the department. Their mean age was 20 (range 5-81), and 429 (68%) were aged 11-20. Seventy one patients were brought to the hospital by ambulance. Most patients (501) were injured as a result of direct contact with the ice or barrier; 75 were injured by twisting or stretching while falling and 49 by direct contact with the blade of a skate; nine presented with blisters caused by ill fitting skates; and one had an acute attack of asthma and one was winded. The table shows the nature of the injuries. The commonest injuries were bruises to the knee (12%) and head (12%) and fractures of the wrist (8%).

Altogether 447 patients had a radiographic examination. Most patients (410) were discharged, 190 were reviewed, and 36 were admitted as inpatients.

The mean weekly attendance at the ice rinks was 14 204 at the larger rink and 2103 at the smaller. The mean proportion of skaters who were treated was 0·16% a week. The correlation coefficient of the percentage of skaters injured against the weekly attendance figures for the rinks was −0·36.

On the assumption that it costs £3.25 per mile for ambulance journeys, £12.60 per patient for basic accident and emergency care, £25.40 per unit for radiology, and £75 per patient day for those admitted to hospital, the estimated total cost to the area health board of treating these patients was £43 276, or £68 per patient.

Comment

During the six months of the study 20 317 new patients were treated in the accident and emergency department. The 636 patients from the ice rinks therefore constituted 3% of the total, compared with 757 (4%) patients who were injured in road accidents in the same period. Only 0·16% of the people attending the ice rinks needed hospital treatment, but this represented many injuries because of the large number of people who go skating. The proportion of skaters injured did not correlate with the number attending the rinks. Thus it is uncertain whether limiting the number of skaters at a rink at any one time would reduce their chance of being injured.

The severity of injuries might be reduced if skaters wore protective clothing. Knee and head injuries, the most common injuries identified in the study, could be lessened by the use of knee pads and helmets; many ice rinks in Canada insist that all skaters under the age of 16 wear head protection.

This study showed that a newly opened ice rink will impose appreciable extra work on the nearest accident and emergency department. The cost of treating these patients is considerable and should be taken into account when new ice rinks are planned.

I thank Mr J Shaw; the clerical, nursing, and medical staff of this department; the management of Dundonald Ice Bowl and Crystals Arena, Bangor; and Mrs V Hastings and Mrs M Onesti for their typing.

1 Horner C, McCabe MJ. Ice skating and roller disco injuries in Dublin. *B J Sports Med* 1984;18: 207-11.
2 Prescott MV. The effect of opening an ice rink on the accident and emergency department of a district general hospital. *Arch Emerg Med* 1986;3:107-10.

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Injuries received at ice rinks

Site	Total No	Nature of injury				
		Fracture	Dislocation	Laceration	Bruise	Strain
Knee	131	4	2 (Patella)	4	79	42
Wrist	96	51		2	4	39
Head	86	2		10	74	
Hand and fingers	64	12	1	37	2	12
Face	49	2 (Nose)		38	9	
Ankle	41	12			2	27
Elbow	32	11	1		20	
Shoulder	26	7	7		12	
Scaphoid	23	1			22	
Pelvis	19	7 (Coccyx)		2	10	
Lumbar spine	11			2	6	3
Foot	11			6	5	
Tibia or fibula	11	9				2
Chest	9			1	8	
Fibula	7	7				
Neck	4					4
Miscellaneous	16					
Total (%)	636	125 (20)	11 (2)	102 (16)	253 (40)	129 (20)